EPILEPSY AND EPILEPTIC INSANITY.

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Part I.

Definition and Classification.

Epilepsy is a disease boasting of a lengthy ancestry and a varied nomenclature. It is described by Hippocrates, who lived in the fifth century, B.C., and its plethora of titles serves to remind us in a great measure of its history and importance. The name in common use - epilepsy_ - takes us back to the time when our forefathers had perhaps a firmer belief in the existence of a spiritual world than we, their descendants, for it was given to this disease because it was thought by them that the condition was due to the interference of evil spirits with

mankind. To what other agency could it be due? To see a person, apparently in good health, fall to the ground insensible; to hear the startling cry accompanying the fall; to look upon the cyanosed countenance and frothy saliva, oftentimes tinged with blood; to behold the purposeless, convulsive movements of the limbs, all these things served to evoke the idea that this could only be the work of a malignant spirit, who seized his victim and hurled him to the ground with relentless fury.

"When, added to the contortions and unconsciousness of the disease during a fit, there were afterwards developed strange hallucinations, terrible acts of impulsive violence, or striking religious delusions, we cannot wonder that a supernatural cause was almost universally believed in of old."[1]*

The afflicted person was the subject of an "epilepsy" - a seizure, from the Greek ἐπιλαμβανειν to seize upon - or its English equivalent - "falling sickness."

Amongst the many names by which this condition is distinguished may be mentioned "morbus sacer", a name bestowed either because the priests of Apollo were epileptic, or because they feigned epilepsy in order the better to impress the clients who came to inquire at their oracles. As the result of an attack of epilepsy

* The numbers refer to the Bibliography on page 82.

was, in early days even as now, a weakening and impairment of the mental faculties, the Romans called it the "morbus sonticus", a legal term signifying exemption from duty, the idea in this case being that the mental faculties were so disturbed as to preclude the presence of responsibility. Another name which is interesting on account of the idea represented by it is "morbus sideratus" - the star-struck disease - for just as it was considered that a lunatic was one who had lost his normal mentality through the untoward influence of the moon; so an epileptic was supposed to have received a blow from the stars, or was affected by the influence of some other heavenly body. Other names given to this disease in olden times were "morbus caducus", that most commonly used; "morbus insputatus", a designation referring to one of the common phenomena of the classic type of the disease, viz: foaming at the mouth, or perhaps because those who witnessed an attack were accustomed to spit upon the epileptic man, or into their own bosoms, either to express their abomination, or to avert the evil omen from themselves; "morbus daemoniacus", a name allied to epilepsy, being, like it, an attempt to describe the supposed cause of the condition, viz: that of being possessed by devils; and "morbus foedus", so given because of the relaxation of the sphinct~~us~~ers, which so often accompanies an attack

of the disease.

In the consideration of this subject it is of the first importance to have a clear idea as to what is usually understood by the condition; in other words, to have a definition, brief enough to be easily remembered, and comprehensive enough to contain the essential facts. It is mostly difficult to frame an accurate definition of any sort or kind, it becomes more so in cases of disease, and especially when, as is the case with epilepsy, the phenomena are so varied. Is there a symptom common to all these phenomena? Or is there any part of the phenomena essential to the condition being recognised as epilepsy?

Let us quote the definitions given by some of the writers recognised as authorities on the subject. Professor Clifford Allbutt says that epilepsy consists of "recurring attacks, sudden and very brief, of disturbance of some of the cerebral functions acting on consciousness, which are not due to a cause outside the brain."

Sir William R. Gowers in his "Epilepsy and other Convulsive Diseases" says:- "The characteristic of the malady is the recurrence of sudden brief disturbance of some function of the brain, varying in degree, extent and character, but generally attended with an arrest of

consciousness sufficient at least to interrupt the control of the muscles necessary for the maintenance of the erect posture."

Spratling points out[2] that epilepsy has two chief forms of manifestation, the one, loss or impairment of consciousness; the other, loss or impairment of motor co-ordination, and also says either of these two forms may be absent. This last statement is apparently in accordance with the opinions of Gowers, who states in the Phonographic Record of Clinical Teaching for Oct. 1905: "It is a common error, due to statements made from the observation of too few cases, that loss of consciousness is an invariable characteristic of the minor attacks of idiopathic epilepsy. Consciousness may be unaffected throughout, but it is more common, in the cases in which it is not lost, for there to be a partial loss, an imperfect realisation of the surroundings, a sense of strangeness for a moment, or some similar feeling which means that the perception of the relation of the individual to his environment is rendered imperfect for a moment by the process which is taking place in the brain." On the other hand, Aldren Turner says[3] that "epilepsy is a paroxysmal convulsive disorder of greater or less intensity, the characteristic feature of which is a sudden loss or impairment of consciousness;" and again:"the feature whose

presence is essential to establish the diagnosis of epilepsy may consist of any degree of unconsciousness, from a momentary blunting up to complete abolition of conscious sensation."

Spratling's definition is as follows:[4] "Epilepsy is a disease or disorder affecting the brain, characterised by recurrent paroxysms which are abrupt in appearance, variable in duration, but generally short, and in which there is impairment or loss of consciousness, together with impairment or loss of motor co-ordination with or without convulsions."

In this definition the loss or impairment of consciousness is evidently considered essential, so that it is hardly in agreement with his previous statement. He seems to regard the epileptic attack as centering round consciousness or mobility, and if neither of these is disturbed the patient cannot be said to have an attack.

There are cases where there is no motor co-ordination, and, says Spratling, there are cases where "patients pass through the most violent and general muscular contractions, during which there is enough consciousness left for the individual to distinguish to some extent what is going on about him, and to some extent remember and tell of it afterwards." Two phrases in the above quotation, viz: "enough consciousness left",

and "to some extent," make it appear that the author recognises impairment (not necessarily loss) of consciousness to be an essential feature of epilepsy, and so he falls into line with the opinions expressed by other authorities. We must therefore regard as a lapsus calami the previous statement that impairment (or loss) of consciousness may be absent without the case losing any of its epileptic character, unless, indeed, the writer is here including Jacksonian epilepsy, in which, as we shall see later, consciousness is often not lost.

While therefore we may have an essential feature of epilepsy, the absence of which would be a reason for disputing the diagnosis, the condition (as has been previously noted) is so varied in its manifestations as to necessitate (for convenience sake) a classification based upon its symptomatology, or the phenomena presented. At any rate, this is the basis for classification usually adopted. Probably the ideal classification of this or any other diseased state would be one based either upon its etiology or pathology, but in the case of epilepsy our actual knowledge of the pathological conditions underlying its manifestations is so meagre, and as the etiology of the disease is largely a matter for speculation, we are driven to regard the symptomatology of epilepsy as the most convenient basis upon which to divide cases into

classes. Not that such classes would differ, except perhaps as regards the position and extent of the cortical area affected (for that the cortical portion of the brain is the part affected is now admitted by all competent authorities), but that for the sake of a clinical description, it is convenient to arrange cases under headings which shall convey to the mind a definite syndrome. The classification offered by Spratling is as follows:-[5]

1. Grand mal, the classic type of an epileptic attack in which consciousness is entirely lost, and motor co-ordination completely destroyed.

2. Petit mal, a less severe type in which consciousness may be only impaired, and motor co-ordination not necessarily destroyed.

3. Jacksonian epilepsy, in which consciousness is not as a rule either impaired or lost, and in which the muscular movements are limited to one limb or to a single group of muscles.

4. Psychic epilepsy, in which the attack affects consciousness only, leaving the physical condition undisturbed.

A very convenient, comprehensive and clinical classification is given by Aldren Turner[6] under the name of eclampsias, or convulsive disorders. He groups together those conditions (often differing from each other

as to their pathology) in which muscular contractions or spasm holds a prominent place. It is comprehensive, for it includes those conditions in which the symptoms cannot be differentiated from essential or idiopathic epilepsy. It includes those conditions, such as infantile convulsions, which in many cases develop in later life into epilepsy, so that "whether we hold epilepsy to comprise every motor spasm, or restrict it to the periodic recurrence of general convulsions begun and accompanied by unconsciousness"[7], there is a place for it to be found in Turner's grouping - which is as follows:-

1	Convulsions symptomatic of primary affections of	A. Nervous System including	A. Partial or Jacksonian epilepsy. B. Convulsions of organic nature.
Eclampsia		B. Urinary System	A. Uraemic convulsions. B. Puerperal convulsions.
2	Infantile convulsions from various causes.		
3	Idiopathic Epilepsy	A. Le Petit Mal (equivalent to Spratling's "psychic" epilepsy.)	
		B. Le Petit Mal with slight convulsions.	
		C. Le Grand Mal.	

Spratling also gives a classification founded upon the etiology of epilepsy. It is rather complicated,

but a simplified form will be found below. The amplified form is, perhaps, cumbrous for practical purposes, but may be found of use from a statistical point of view. Taking as a basis the incidence of age, he makes three chief divisions and groups the causation round each of them.

1. Age to 3 years - infantile inherited, accidental, traumatic and idiopathic.

2. Age between 3 and 20 years - accidental, traumatic, developmental and idiopathic.

3. After 20 years - accidental, toxic, traumatic, idiopathic and senile.

We shall follow Spratling's classification (vide supra p.8) based on symptomatology, for it is a simple, definite, and comprehensive one; it is also pathologic as far as our present knowledge permits it to be. For while we are to a great extent ignorant of the actual conditions obtaining in the nerve cells of the cortex which eventuates in the production of a fit, it is agreed that the cerebral cortex is the portion affected. In the severer forms it would appear that the entire brain cortex is affected, in the less severe forms, petit mal with slight spasm and Jacksonian epilepsy, the lesion remains localised, while in the purely psychic attacks, the frontal lobes are specially implicated.

It does not follow that attacks are confined to

one or other of the forms mentioned above. I have seen a patient who at one time would have an attack of grand mal, at another an attack of petit mal, while a third attack was of the psychic type.

Part II.

Causation and Pathology.

The causes of epilepsy may be considered under two heads - predisposing and exciting. Of the predisposing causes, heredity undoubtedly comes first. Gowers is distinctly emphatic on this point - Epilepsy is an inherited disease,[8] and other authorities speak with equal certainty. "In epilepsy," says Voisin,[9] "it plays the chief rôle." Does this mean, then, that epilepsy as a morbid entity is transmitted from parent to offspring? No, for epilepsy is not a morbid entity, says Echeverria. It is a symptom-complex which may, according to Féré, be due to very different pathologic conditions.

The meaning of heredity as a predisposing cause of epilepsy lies in the fact that a child who is the product of epileptic ancestry is born with a faulty nervous system, the integrity of his nerve cells is already impaired, and they are less able to withstand the untoward influence of any exciting cause. It need not even be the case that epilepsy itself should have existed in the child's progenitors, the existence of other neuroses, as for example, one of the numerous forms of insanity, is sufficient to determine the predisposition of the child to epilepsy.

Gowers says[10] "Many persons seem to have a congenital instability of nervous tissue, increasing with its development and constituting what we call a predisposition to epilepsy, and this is more common in members of families in which there is some such tendency, although it may lead to disease only in a few and rare instances."

In order the better to understand the rôle that heredity plays in the production of epilepsy, it is of importance to have a clear idea of the supposed changes underlying the epileptic condition. The importance of this is also seen when we consider what has been said concerning the etiology of the disease, for our ability to recognise any other diseased state (e.g. scarlatina) as a factor in the production of epilepsy must surely depend, at any rate to a great extent, upon the idea we have formed as to the nature of the pathological changes taking place in the brain cortex of the patient. Still further, the importance of a clear idea of the pathology of epilepsy will be readily understood, when we consider that the scientific employment of therapeutic measures is, or should be, based on pathology. Why, for example, do we prescribe the bromides? Is it merely as an experiment in empiricism? It has been established beyond doubt that epileptic attacks become fewer and sometimes entirely cease under the action of the alkaline bromides - are we

able to give an intelligent reason for this result? Must we say - we see that such a result has occurred, but why - we know not? If we can correlate the known medicinal action of the bromides with out theory of the epileptic state, our treatment of the condition will, at any rate, be lifted out of the rut of empiricism.

It is agreed on all sides (as already said) that whatever the ultimate lesion underlying the epileptic phenomena, its locale is in the cerebral cortex, in the gray matter of the brain. Now the function of the cells in this gray matter is to store up nervous energy, to be used as required. How this nervous energy is obtained, that is to say - by what process of metabolism it is acquired and in what manner it is stored up in the complexities of the cellular elements - are interesting questions, which however we do not propose to discuss in this connection. Suffice it to say, that energy <u>is</u> stored up, and that it is gradually discharged to meet the demands of the organism. Surely it seems but common sense to admit that the regular normal storage and regular normal discharge must be dependent upon the structural integrity and due nutrition of the nerve cells, and that anything tending to impair this integrity and alter this nutrition must disturb the equipoise of storage and discharge existing in the cells of a normal brain. In the normal

cell the balance between storage and discharge is probably due to a power of inhibition possessed by the cell, and if the integrity of the cell be impaired, or the conditions of its nutrition altered, it is but reasonable to suppose that nervous energy will be discharged irregularly and spasmodically.

If this be so, it is not difficult to understand how important is the question of heredity as a predisposing factor in the production of epilepsy. In a person of neurotic type there is every reason to believe that the offspring will be ushered into the world with an ill-developed central nervous system, with cells ill-equipped for their function under even normal conditions, so that when there is the slightest deviation from the normal conditions in the direction of stress, the equilibrium is disturbed, and the sudden release of energy occasions a "nerve storm."

It has already been observed that by heredity is not, of necessity, the existence of epilepsy in the ancestry, but of any neurosis, or of any condition likely to cause degeneration of nerve tissues.

Before passing on to consider the exciting causes of epilepsy, it is clear that attention should be given to its pathology, inasmuch as our conclusions with regard to the one must be influenced by our belief with respect

to the other. If we have no definite idea in our minds as to the determining cause within the brain for the production of a nerve storm, we cannot reasonably take up the position that any systemic condition can be said to contribute to this cause. Why, for instance, should we assert that the convulsions of dentition are an exciting cause of epilepsy, unless we can trace the pathway from these to the ultimately impaired nerve cell in the cerebral cortex?

Now, in considering the pathology of epilepsy, we are at first non-plussed by such a statement as this:- "The naked eye examination of the brains of epileptics is, as a rule, negative. No constant pathological lesion has been detected ... Notwithstanding the advances made within recent years into the minute histology of the nerve cell, the morbid change - if any - underlying the epileptic condition is still undescribed.... What is the cause of a periodical excitability of this (the cortical) region, or whether any change other than a hypothetical molecular one occurs in the nerve cells, is at present unknown."[11]

Most of the naked eye appearances found in epilepsy have no intrinsic value as far as the malady itself is concerned. They are multitudinous, and include haemorrhage, thrombosis, embolism, sclerosis of various parts

of the brain, such as the cornu ammonis, thickened meninges, (the dura often being found adherent to the skull) osteo-sclerosis, etc. Many of these are of importance only in proportion to their tendency to produce an unstable organism, some are the accidental results or concomitants of the fits, while many are associated with diseased states other than epilepsy.

As the seat of the changes is in the cortex we must consider what modifications might give rise to epilepsy. Undetermined and undemonstrable molecular changes in the nerve cells together with gliosis of the cerebral cortex is the reason given by Gowers and Jackson. That there is some chemical change in the metabolism of the nerve elements resulting in a product of a toxic character is the theory adduced by some. Others have pursued this idea of toxic elements being present not only in the nerve cells, but also in certain of the secretions and excretions of the patient. Others, again, have attempted to fathom the secret by observations directed to the nature of the fit, and the part played by motor and sensory elements in the production of the convulsive phenomena.

The experiments of Prus[12] were devoted to determining the origin of the fit. He divided the pyramidal tracts on both sides at various levels from the internal

capsule to the cord, and on stimulating the cortex found that there was no change in the development or generalisation of the attack. His conclusion was that the generalisation of the fit did not take place by means of the pyramidal tracts. A confirmative experiment was to cut entirely through the peduncles, except the pyramidal fibres, and those of the pons varolii, and he then found that no fit could be produced by electric stimulation of the cortex. His conclusion was that the motor impulse was transmitted by fibres other than those of the ordinary motor paths, and that these fibres were to be found in the dorsal portion of the peduncles. Prus then endeavoured to ascertain where the origin of the epileptic spasm was, whether it was in the motor or sensory elements of the cortex, and he came to the conclusion that it was either in "the end tufts of sensory nerves in the cortex, or in the sensory cells of the cortex and not in the motor cells or fibres." Having arrived thus far, the next question was - What particular sensory elements in the cortex were concerned in the origination of the epileptic attack? Prout endeavours to answer the question from a careful microscopical examination of the cortex of patients dying during a period of status epilepticus, and states that it is the second cortical layers in which the cells present

changes of a striking character. He says:"The cells are swollen in many instances to twice their normal size, the nucleus being especially large and granular, with indistinct outline. The limitations of the nucleus are often difficult to determine, the chromatic substance has almost disappeared from the body of the cell, and this portion of the cell appears ragged and poorly outlined. The most striking changes are found in the nucleus. In addition to being granular, swollen and poorly outlined, the nucleolus is often absent, having been abstracted from the nucleus in the process of section making."[18] Prout deems this last fact of exceptional importance, inasmuch as the "formative power of the cell centres in the nucleus," that it is the "especial organ of inheritance," that "it plays an essential rôle in chemical synthesis," that "digestion and absorption of food and secretion cease with its removal from the cytoplasm," and that "fragments of protoplasm deprived of the nucleus die." It might, in passing, be observed that the findings of Bevan Lewis in cases of epileptic insanity are similar, namely, that there exists fatty alteration in the nuclei of the cells of the second cortical layer, with vacuolation and disintegration of cell protoplasm.

It has already been stated that one group of

observers considered the origin of the epileptic fit to be due to a pronounced cerebral gliosis which was accompanied by fine molecular changes. It would appear, however, that the gliosis, which is indeed found in so many cases of epilepsy, is the consequence of previous changes in the cortex rather than the cause of the epileptic condition. Granted that the above statements are correct with reference to the destruction and disintegration of the cellular elements of the second cortical layer, it is not difficult to see how nature endeavours to fill the void by an overgrowth of neuroglia. Nerve cells once destroyed are not renewed, but neuroglia is a proliferating tissue, and as there is probably an active process at work, namely, that of a toxin, acting directly upon the neuroglia and causing it to proliferate in response to the irritation of the toxin; so that the space left by the disintegrated nerve cells is also filled up by the neuroglia, - this is sufficient to account for the hyperplasia observed as stated above.

The conclusions drawn by Prout and Clark after a careful discussion of the views held by others, and based upon experiments of their own, are as follows:-

"Epilepsy is a cerebral disease attended and followed by profound and diffuse cortical degeneration.

The morbid changes concern chiefly the destruction

of the nuclei of the cells of the sensory type from which the primary departure of the disease originates. Its terminal pathology is a progressive gliosis more or less marked and diffuse.

Epilepsy is essentially a sensory phenomenon with a motor manifestation.

Its etio-pathology rests with a variety of toxic or auto-toxic agents not as yet definitely isolated or determined.

The disease is engrafted upon a cortical organic cellular anomaly which is induced largely by a faulty heredity, the exact anatomic nature of which is not known."[14]

This gives us a clear and concise summing up of the pathology of epilepsy as far as is known at present.

Having, therefore, these findings before us, we may now retrace our steps somewhat, and consider further the etiology of the disease, and endeavour to ascertain whether those bodily conditions which have been thought to stand in causal relationship to the disease, can be said to result in the pathological changes which have been noticed.

The influence of heredity has already been alluded to, and the existence of nerve cells of impaired integrity either of structure or function, or most

probably both, has been seen to lie at the basis of the epileptic phenomena.

But heredity is not the only cause of epilepsy. Even in those cases where no other cause can be discovered, it is more than possible that an excitant has been at work somewhere, - some form of stress, perhaps, disturbing the normal equilibrium of the structural nervous elements.

Leaving, then, the question of predisposing causes, we come to the consideration of exciting causes, and possibly the commonest cause of epilepsy in early life is haemorrhage. Spratling states as the result of his observations on this question that 11 per cent had epilepsy as the consequence of this cause. These haemorrhages (which sometimes produce paralysis) have their location in the bloodvessels in the neighbourhood of the central motor neurons. They may be pre-natal or post-natal. When occurring during intra-uterine life they are often due to some injury received by the mother during pregnancy, or to some injury inflicted on the child in instrumental deliveries. Hughlings Jackson was of opinion that the commonest cause of idiopathic epilepsy was small haemorrhages in the brain. And it is important to bear in mind that the size of the haemorrhage does not necessarily bear any relation to the intensity of the

epileptic fit. The size of the lesion is of far less importance than its location. When haemorrhage occurs after birth there must be some internal cause for it, excluding now those cases that are admittedly of traumatic origin. We have seen that haemorrhages in the brain will cause the convulsions of epilepsy, but may we not also say that convulsions may produce haemorrhages as a consequence of the intense cerebral congestion accompanying them? And if this be so, we might proceed a step further, and ask to what might the convulsions be due?

They may be due to numerous causes, such as - improper food and imperfect digestion, and this brings us to the place occupied by dentition in the production of epilepsy. And whereas it is usual to ascribe many infantile ailments to the "teeth" as a matter of course, and while also it is difficult to determine with exactness the causal relationship between dentition and epilepsy on account of the numerous other causes often existing at the same time capable of inducing convulsions in children, yet the foremost authorities on the subject agree in assigning to dentition a position of great importance in the etiology of epilepsy. Spratling[15] says that "dentition when severe, and acting on an organism that bears the impress of transmitted weakness, plays an important rôle in the production of epilepsy in early

life." Gowers says that in at least one-tenth of the cases of epilepsy a relation to infantile convulsions (not necessarily due to teething, however) continuous or intermittent, can be traced. Smith says[16] "Among the more common pathologic results of difficult dentition are certain affections referable to the nervous system. Eclampsia is one of the admitted results." Barrier attributes convulsions in the teething infant to excitement of the nervous system, arising from pain which is felt in the gums and to a determination of blood in the dental apparatus, in which reflex the vascular system participates. Difficult dentition must be considered not so frequently a direct as perhaps a predisposing cause, producing a sensitive state of the nervous system, or possibly an afflux to the head, and which by an additional trivial stimulus ends in convulsions. Dr Clouston remarks:- "I have seen the convulsions of dentition followed by prolonged delirium, ending in idiocy, or in true epilepsy, or insanity of adolescence." Other writers deny that convulsions are caused by dentition alone, so that probably the truth of the matter lies in the statement that difficult dentition arising in a constitution already weakened and retarded in development by hereditary disposition or acquired conditions, excites reflex convulsions which produce haemorrhages, the result of

the intense cerebral congestion, these being antecedent to the true epileptic fit. But how does this question of haemorrhage harmonise with our pathology of the subject? Is it that the cells of the sensory layer are deprived of their nourishment on account of the foci of softening around them occasioned by these haemorrhages? Is it that in consequence of this loss of nutrition the integrity of the sensory nerve cells becomes impaired, the cell itself degenerates, and has no longer an inhibitory influence over the motor cells?

We turn next to the infectious fevers as factors in the production of epilepsy. Spratling gives scarlet fever as the cause in 25 out of 1,323 cases, or a little over 5 per cent, and considers that quite 2 per cent of all cases arise from this cause. It is noticeable that usually the convulsions which eventuate into epilepsy do not occur during the active stage of the fever, but afterwards, and commonly in association with nephritis. Gowers agrees with the statement that scarlet fever is often the cause of epilepsy, and considers that the fits are due to the action of the poison of scarlet fever upon the nervous system.

With regard to whooping-cough as a cause of epilepsy, Spratling gives 1 per cent of all the cases that have come under his observation, and ascribes the

convulsions as being due either to some local irritation, as a thrombus, embolism, or haemorrhage, or to the action of toxic products circulating in the system. Other infectious fevers, as diphtheria, measles and malaria have also been credited as being the cause in occasional cases.

It is not at all uncommon to be told that the origin of a fit was a fright. That this idea has substantial foundation appears from the proportion of cases collected by Gowers, who states[17] "that of all the immediate causes of epilepsy the most potent are psychical, - fright, excitement and anxiety, to these are ascribed more than a third of those in which a definite cause can be given. Of these three forms of emotion fright takes the first place. The relation of this cause to age is, however, very distinct. It is effective chiefly in early life when emotion is so readily excited, and is most powerful at the transition from childhood to adult life, while after middle life it is almost inactive. Of 173 cases only 14 commenced after thirty years of age, and 145 began under 20. Of these the majority, 102, began between ten and twenty, only 43 cases before ten. The female sex is notoriously the more emotional, and accordingly the disease results from fright in a larger proportion of women than of men, although the

difference is, perhaps, less than might be expected, 61 per cent of women and 39 per cent of men. It is notorious also that this difference between the sexes increases as life advances. In childhood one sex is almost as emotional as the other, but with puberty men become far less emotional than women. The influence of fright as a cause of epilepsy is in accordance with this fact. Under ten years of age the sexes suffer equally. Between ten and twenty the male sex suffers less than the female sex, as 3 to 4; between twenty and thirty, as 3 to 13; and over thirty the only cases due to this cause occur in women. Predisposition usually exists. Hence in speaking of fright as a cause of epilepsy it must be remembered (as already stated) that its effects are only that of the the exciting spark."

Spratling agrees with Gowers in regard to the great importance of fright and other emotions as a cause of epilepsy, but differs from him in that he considers the most potent cause to be the cerebral palsies of early life rather than fright, a physical cause rather than a psychical one. Whether emotional causes resulting from the sight of another in an epileptic fit is sufficient to induce an attack is doubtful. Sir Thomas Watson says:-[18] "There is yet another and singular occasional cause of epilepsy that deserves to be mentioned, namely, the sight

of a person in a fit of that disease." He also states that patients who have never had a fit may acquire the disease in this way; and indeed, if Gowers is right in attributing to fright and other emotional causes such an important place in the etiology of epilepsy, it is difficult to see why the emotional state occasioned by the sight of an epileptic should not have the same effect as a fright induced by other causes. Particularly is it feasible that this might be the case if the fit is seen for the first time, and if the nervous system of the individual is already in a condition of instability through hereditary disposition. Amongst the emotional causes may be included any form of stress - anxiety, grief and overwork, which seem to loom so largely in the production of other forms of nervous disorder. In such cases, the sudden stress upon the nervous system serves to destroy the balance of equipoise of the motor and sensory elements by disturbing the process of nutrition in them.

Coming to trauma as an alleged cause of epilepsy, there are two points of interest to be considered. One is whether these "fits" due to trauma are epileptic in the beginning; and the second is that in these cases a hereditary predisposition need not be present. With regard to the first Spratling is inclined to answer in

the negative. He says[19] "the convulsions due to trauma are of the Jacksonian type, appearing locally in one arm or leg, or in one side of the body, because the injury is local, affecting as a rule a limited portion of the cortex, generally in or near the motor region." It does not always follow that the cortical lesion can be demonstrated. In support of this he refers to a case of a woman in whom the convulsions began locally, namely, in the right thumb, then the hand was affected, after that the fore-arm, the whole arm and right side of the body. She died in status epilepticus, after having had about 200 attacks. A careful microscopic study of numerous sections of the brain from the right thumb centre showed no lesion whatever, beyond a condition of cell vacuolation, that we should naturally expect as the result of extreme exhaustion.

But there seems no reason not to include this under the heading of true epilepsy. Trauma would cause haemorrhage, and if idiopathic epilepsy is the result of "small haemorrhages in the brain" (Hughling Jackson) due to internal causes, why should it not also be induced by traumata or causes from without?

With reference to gastro-intestinal disorders in the etiology of epilepsy, the rôle of these is of great importance. We have already referred to the fundamental pathological principles of the impaired nerve cells in the

cortex of the epileptic as an acknowledged fact. "The disease," says Prout, "is engrafted upon a cortical organic cellular anomaly which is induced largely by a faulty heredity, the exact anatomic nature of which is not known. - And again:"Its etio-pathology rests with a variety of toxic or auto-toxic agents, not as yet definitely isolated or determined." Bearing these conclusions in mind, it will not be difficult to understand the part played in the production of epilepsy by such conditions as gastro-intestinal disorders, and toxic agents such as lead; toxic conditions such as that of scarlatinal nephritis; pregnancy and the puerperal state in which the quality of the blood is altered.

If the presence of toxic or auto-toxic products is acknowledged as an important factor in epilepsy, it will be seen that any condition capable of giving rise to a toxic state of the blood is prima facie a possible cause of the disease, because they (the toxins) circulating in the bloodvessels of the cerebral cortex impair the integrity of the nerve cells by interfering with their nutrition. "In health," as Gowers remarks, "energy is liberated in response to a definite stimulation. Such capacity for instant action involves a delicate equipoise of the processes for the liberation of nerve force and for its restraint and control. The balance must depend

on the process of nutrition in the nerve structures, for the liberation of energy depends on the occurrence of chemical processes under the influences of life - processes which must ever be on the verge of destruction." If the cells are properly nourished, then the equilibrium is maintained, but if not, it is impaired or destroyed. It is necessary for the maintenance of this equilibrium that the normal processes of nutrition of the cells be undisturbed. This disturbance may be affected by altering the quality or character of the material which is carried to the cell, and so long as the food taken is of a suitable nature, and the assimilative processes normal in character, so long will there be a maintenance of cell-nutrition, but let there be from any cause an alteration in the character of the material so as to render it incapable of supplying due nutrition to the cellular nerve elements, then we have conditions resulting in impairment of the cell and ultimately, it may be, in its disintegration. It may be said therefore, that whatever condition, whether of body or mind, that is to say physical or psychical, is capable of producing these changes, may be regarded as a possible exciting cause of the epileptic state, its importance in the rôle of etiology being in direct ratio to the degree and intensity of its action.

In the British Medical Journal for March 3rd. 1906,(p.496)

there is a very interesting account by Dr. John Turner of his experiments with a view to determining the causation of the fits. His theory is that "epilepsy is a disease occurring in persons with a defectively developed nervous system associated with a morbid condition of the blood, whereby it shows a special tendency to intra-vascular clotting, and that the immediate cause of the fits is sudden stasis of the blood stream resulting from the blocking of cerebral vessels by these intra-vascular clots."

After describing the histology of the defectively developed nerve cells, Dr. Turner proceeds to describe experiments bearing on the relationship between stasis of the cerebral circulation and convulsions, and shows that convulsions can be set up by blocking the cerebral vessels. He then points out that in 90 per cent of epileptic brains examined, intra-vascular clotting had taken place. This clotting (he holds) is due not to the condition of the vessels, but to the state of the blood. He believes also that in the cases of epilepsy where cortical or meningeal haemorrhages had occurred, they were due to rupture of the vessels in consequence of a clot having formed.

Anticipating two objections, first, that the thrombus might be the result and not the cause of the fits; second, that the clots might have formed during a moribund condition of the patient - Dr. Turner points out that

clots occurred in great quantity in patients who had no fits for a long time before death, also that the presence of phosphorus in the clot was evidence (in his opinion) that the clots were not post-mortem.

Some points in favour of this theory which may be mentioned are - the frequency of epileptic fits at night when the blood pressure is low, and stasis more likely, also the fact that certain diseases and states in which the blood is more fibrinous and therefore more coagulable, such as scarlet fever, and perhaps typhoid, ~~and~~ also the puerperal state, have special importance in the etiology of epilepsy.

But assuming the correctness of the theory, the question has yet to be answered:"What are the special conditions that determine a periodical intra-vascular coagulation?"

Part III.

Symptomatology.

Reference has been made on a previous page to the different types of epileptic fits by which a classification may be made for clinical purposes, viz: Grand mal, petit mal, Jacksonian epilepsy and the purely psychic form. The first mentioned is the classic type, and it is not intended to give in minute detail a description of it, but merely to refer to several points of interest in connection with it. It may, nowever, not be without interest to quote a poetic description by Lucretius written some two thousand years ago, in which we have a graphic account of a 'grand mal' seizure, differing in no wise from the clinical aspect of the attack as presented to us in the present day, and which might with equal truth have been composed by a poet of the present century, instead of twenty centuries ago.

In these lines, as will be seen, we have the main features of the classic attack, with the exception of the aura, viz: the sudden onset, the fall, the cry, the loss of consciousness, the convulsions (tonic and clonic) and the stertorous breathing.

"Oft, too, some wretch, before our startled sight,
Struck as with lightning, by some keen disease,
Drops sudden: By the dread attack o'erpowered
He foams, he groans, he trembles and he faints;
Now rigid, now convulsed, his labouring lungs
Heave quick, and quivers each exhausted limb.
Spread through the frame, so deep the dire disease
Perturbs his spirit: as the briny main
Foams through each wave beneath the tempest's ire.
He groans since every member smarts with pain,
And from his inmost breast, with wontless toil,
Confused and harsh, articulation springs.
He raves since soul and spirit are alike
Disturbed throughout, and severed each from each
As urged above distracted by the bane.
But when at length, the morbid cause declines,
And the fermenting humours from the heart
Flow back - with staggering foot first treads,
Led gradual on to intellect and strength."

Attacks of grand mal are by far the commonest form of epilepsy, the proportion being, according to Spratling, about 60 per cent. Then come in order attacks of petit mal, Jacksonian epilepsy, while cases of a purely psychic type are least common of all. It is to be remembered,

however, that epilepsy is not bound to "fixity of type", it often happens that not only may attacks of petit mal be followed by attacks of the classic type, but that attacks of grand mal, petit mal and psychical epilepsy may be interchangeable in the same patient. The rule is that lighter attacks develop into the more severe.

The epileptic fit is often ushered in by an aura, which therefore serves as a warning to the patient when occurring at an appreciable interval before the attack. The word signifies a "vapour or emanation from a body, surrounding it like an atmosphere." It is said to have been first used by Pelopis, master of Galen, who thought that the fit began in the form of a spirituous vapour in the veins of the extremities and ascended to the head, whereupon the patient became unconscious. Aurae are of many kinds, - but they may be conveniently classified under four heads:- (1) Sensory, those that cause some disturbance in the sensation of the patient; (2) Psychic, those associated with some mental disturbance; (3) Motor, when the muscular system is involved; and (4) Irregular, or mixed, occurring in those cases that cannot be altogether classified under either of the other three captions. Gowers' classification of the aurae is somewhat more expansive, as he divides up the sensory aurae thus:- (1) Unilateral, and (2) general aura, such as sensations in the limbs, tremors, faintness, etc;

(3) aura referred to certain organs, especially those supplied by the pneumogastric nerve, most of the visceral warnings coming under this head; (4) vertigo and allied sensations; (5) certain sensations in the head, pain, etc.; (6) special sense warnings.

The most common of all the aurae are those of the sensory class, and of the special sensory aurae those of the visual organs greatly predominate. Their forms are various, sometimes they appear as flashes of light, sometimes as pictures. The aura in one patient under my care saw pictures of animals, tigers, pass before her eyes; another invariably sees "blue birds," flying about in the air; sometimes different colours are seen, and there is often present macropsia and micropsia.

Auditory aurae are much less frequent than visual. In such cases the patient hears noises when all is still, and voices when no one is speaking. Aurae of smell and taste seem to be frequently associated. The patient mentioned above used to complain of an offensive smell in the room, and at the same time of a"nasty bitter taste" in the mouth.

Olfactory aurae are often associated with a peculiar dreamy state. A very interesting case was reported[20] by Drs. Hughlings Jackson and Purves Stewart which shewed that the patient had attacks of epilepsy with a warning of a crude sensation of smell, and with the intellectual aura

or dreamy state. A diagnosis was made of gross organic disease in one tempora-sphenoidal lobe, and as there was right-sided optic neuritis it was suggested that the right lobe was affected.

The epigastric aura is said to be the commonest of all sensory aura. Spratling gives (p.232) 15 per cent as the proportion in the cases recorded by him. The patient complains of a "gnawing" sensation at the pit of the stomach. This aura is believed by Gowers to be connected "with some disturbance involving the centric origin of the vagus."

We pass on to the consideration of those aurae which are connected with some disturbance of the mental condition, namely, psychic aurae. These may be present as in the form of some definite mental picture, or in the form of some emotion or idea. Patients are conscious of "something going to happen," there is an ill-defined dread of apprehension, a confused mental state in which looms large the idea of impending disaster.

Motor aurae are by some considered to be not so much warnings of an impending attack, but actually part and parcel of the attack itself. I have seen a patient running up and down the ward immediately before the actual onset of unconsciousness. Other patients will turn round and round in the place where they are standing, until unconsciousness ensues.

Irregular aurae, as already stated, are those in which there is a combination of two or more of those already mentioned, viz: the sensory, motor and psychic types.

The importance of the aura varies according to its type. If the warning is of the sensory type, and particularly referable to one of the special senses, e.g. sight, hearing, taste or smell, it is an indication of the locality of the cortical area of the brain in which the epileptic discharge commences. A visual aura would point to a lesion in the occipital lobe, auditory sensations to a lesion in the first temporal convolution, while the uncinate gyrus is, according to Ferrier, the centre for taste and smell. Motor aurae, especially if commencing locally, e.g. in the arm, face or leg, point to a lesion in the corresponding areas for these movements in the cortical region adjacent to the Rolandic fissure.

The epileptic cry is often absent. When present it is apparently due to the fixation of the chest by spasm of the intercostal muscles and diaphragm, and is scarcely, if ever, verbal.

The attacks called petit mal, with evident spasm, or epilepsia mitior, are only distinguished from those of the classic type by their diminished severity. There is less muscular disturbance, compared with that in grand mal, and though the mental faculties are also less involved

during the time of the attack, there is greater permanent mental impairment as a result, so that the liability to a terminal dementia is greater in the subjects of petit mal and psychic epilepsy than in those of grand mal. In the psychic form we have no motor disturbance whatever, the mental faculties only are affected, and these sometimes only for the briefest possible period; there is a loss or impairment of consciousness, it is true, but it is so slight as sometimes to be quite unnoticed. Spratling pertinently observes[41] that "in the absence of any knowledge of the mechanism by which the conscious life of the individual is suddenly snuffed out, without even the appreciable tremor of a muscle infinitesimal in size, and of the manner in which it is as suddenly restored, we can at this time only marvel as at something we do not understand."

In what is called Jacksonian epilepsy we have a type differing in many respects from either of the foregoing. Its distinctive features are localised convulsions; (localised at any rate at the onset of the fit, though they may afterwards become general on the side on which the spasm commenced.) presence of consciousness; and in the fact that the attacks are always followed by a temporary paralysis of the parts involved in the spasm.

The convulsions are limited to one half of the body; they may even be confined to an arm or a leg, or even to a single group of muscles. For this reason, this form

has been called partial epilepsy; partial, that is to say, not as referring to the absence of some of the important manifestations, but as referring to the limited area to which the convulsions are confined. The causes assigned for the production of this type do not differ from those credited with the production of idiopathic epilepsy, except that trauma and syphilis seem to play a more important rôle in the former than in the latter. But whatever the assigned cause, it is understood that there is in the cortex cerebri, or in the tissue immediately subjacent, a definite focus of irritation which accounts for the localised form of convulsion occurring at the commencement of the attack. It is evident, too, that the situation of commencing convulsion is determined by the precise location of the lesion in the cortex.

The epileptic cry and aura are as a rule absent in Jacksonian epilepsy.

Part IV.

Epileptic Equivalents.

The two classical types of epilepsy are le grand mal and le petit mal, but besides these there are other clinical phenomena sometimes met with in epileptics, at other times as independent symptoms, which stand in close relation to the psychical condition underlying epilepsy. These phenomena are termed epileptic equivalents. By epileptic equivalents are designated certain states of consciousness "differing not only from normal consciousness, but from the state of consciousness that usually occurs in epileptic attacks." (Peterson) The words themselves imply the existence of a psychical state that takes the place of epilepsy. It is the opinion of some that these clinical phenomena (of which we shall now speak) are not in this sense "equivalents", i.e. taking the place of epilepsy, but that they are rather the sequelae of epileptic attacks which have not been noticed. Turner, however,[52] says:"It is therefore necessary to distinguish between post-paroxysmal psychoses and mental phenomena following convulsive seizures, and phenomena taking the place of ordinary fits." If these clinical phenomena are epileptic equivalents, they cannot at the same time be classed as post-epileptic phenomena,

having regard to the admitted meaning of the word. And what are these phenomena? What are those states that have been considered by authorities to come under the head of epileptic equivalents, and what conditions are necessary to be observed so as to enable us to place them under this head? In answer to the first question, we may say that like epilepsy itself, the equivalent manifestations may be either motor or sensory in character. In the one case there may be a violent outbreak, varying in intensity and duration, in the other there may be no such manifestation, but consciousness as to the environment of the patient is lost, and his actions during this period are altogether automatic. Apart from these two chief forms of equivalents, there are other clinical phenomena believed by some to come properly under this head, but in many other instances it is difficult to come to any definite conclusion, the cases being few and the evidence meagre. For instance, Spratling says (p.177) "Among the visceral epileptic equivalents may be mentioned angina pectoris, which may precede, coincide with, or follow a regular attack of epilepsy." It seems to me that here the author runs away from his original position, and treats as an equivalent what is really an accompaniment, and possibly having no causal relationship whatever with the epileptic state.

Similar remarks may be made concerning asthma,

pertussis, diaphragmatic and laryngeal spasm. It is different with such states as nightmare, narcolepsy and somnambulism and even migraine, which are dependent on cerebral conditions.

With regard to narcolepsy, or periodic falling asleep, Turner gives a case in which the attacks were interchangeable with petit mal seizures. I myself have also seen a similar case, and as in the case mentioned by Dr. Turner, so there was in this case a family history of epilepsy.

It is considered by several authorities that the migraines belong to this group. Women suffer most from this affection, and Spratling believes that some of the lighter forms of epilepsy pass for sick headaches. To diagnose migraine as an epileptic equivalent (and the diagnosis is often very difficult) our attention should be directed to ascertaining the frequency of attacks of periodic headache in the ancestry, also to find out if patients subject to migraine have (at other times) attacks of idiopathic epilepsy, also whether the attacks of migraine have been followed by, and apparently substituted for, true epilepsy. Among other prominent sensory equivalents are the gastralgias and enteralgias; also nausea and vomiting which appear to be incomplete visceral aurae. To decide in any individual case whether the conditions mentioned can be properly

classed as epileptic equivalents, we should endeavour to discover the presence or absence of genuine epilepsy; and whether the visceral attacks come on suddenly without known gastric cause; whether they are periodic, and of what duration. And this brings us to the second question. What general conditions are necessary to justify us in regarding motor or sensory phenomena as epileptic equivalents? Our investigation in this direction should run upon three lines:- We should inquire into (1) the family history of the patient, (2) the personal history of the patient, and (3) the special characteristics of the clinical phenomena presented by the patient. The points to be noticed in this latter connection are the suddenness of onset, brevity of duration and periodicity of occurrence.

I have had under my care a patient who presented several of these epileptic equivalents. Her family history was not good, a sister was subject to fits in childhood, and another sister was neurotic and given to somnambulism. She herself (now aged 22) can remember being "dazed" often while at school. I had daily opportunities of observing her for several years. During the earlier portion of the time, being about 17, she often walked in her sleep, going downstairs, opening the front door, and even (on one occasion) promenading in the garden, though usually I heard her going down the stairs and hindered her further progress.

She was quite unconscious of all that happened during these excursions, and would stoutly deny that she had ever got out of bed during the night.

On the occasion of the walk in the garden the muddy aspect of the sheets convinced her. She was subject to attacks of pain in the epigastrium with nausea and vomiting, and was for some time treated for gastric ulcer. There was, however, never any trace of blood in the vomit. She would go to sleep for several minutes at a time without there being any apparent reason, such as overwork or fatigue, for so doing. About a year ago, owing, as I believe, to a time of stress and anxiety in the household, she developed attacks of grand mal, which became very frequent, and were interchangeable with attacks of petit mal with spasm of the muscles of the left side of the face. She was put on bromides and went home for a rest. She was advised to do without salt in any form. The attacks became less frequent, and when last I heard, she had, according to her own account, been free for three months. Of course, this freedom would be from major attacks. Whether there had been any seizures of a minor character, or any merely psychic spells, it is impossible to say, as my past experience of the patient with respect to these lesser attacks warrants me in saying that she would be ignorant of their occurrence. But, on a review of this case, I am of opinion

that the gastric disturbances, the somnambulism, the narcolepsy, were all epileptic equivalents, and may be properly classified as such. It was not until the major attacks developed that the diagnosis was confirmed as one of epilepsy, several occasions on which she had fallen to the ground (apparently unconscious) having been attributed to the intense pain attendant upon her menstrual periods.

Part V.

Epileptic Insanity.

It has been calculated that ten per cent of all epileptics ultimately become insane, and with reference to those who cannot legally be so certified, it must be admitted that they generally become deteriorated mentally, their mental faculties become blunted, their power of memory impaired. In fact, it may be taken for granted that every true epileptic attack leaves the patient in a greater or lesser degree weak in mind, even if only temporarily. Such weakness is not necessarily to be measured by the severity of the convulsions attending the attack. It does not follow that motor convulsions in an intense degree will eventuate in an impairment of mental faculties to a similar degree. The facts point the other way, and it is generally agreed that those attacks which are purely psychic, or psychic attended with slight muscular disturbance, are more liable to be attended with mental impairment and deterioration than attacks of grand mal. The phenomena accompanying epileptic insanity are so many and varied that it is somewhat difficult to make a satisfactory classification of them. The usual classification, and perhaps the best, is, first to make a classification on the basis of point of time with reference to the occurrence of the fit, and then to

differentiate between the phenomena presented. Following out this idea, we might tabulate as follows:-

1.	Interparoxysmal mental states due to epilepsy. Cases of idiocy or imbecility being the result of epileptic fits during infancy would come under this head.		(a) Dementia - varying in degree from a mere dulling of the intellect to idiocy. (b) Manio-melancholic conditions, with or without hallucinations and delusions.
2.	Paroxysmal states due to epilepsy.	(a) Pre-paroxysmal	mania melancholia
		(b) Post-paroxysmal	mania automatism.

1. The interparoxysmal state should be regarded as the typical epileptic mental attitude. It is the habitual level to which the epileptic is reduced from his former state of mentality. I have called this state one due to epilepsy, but whether it should be so considered, or whether it is part of the diseased condition itself, is a question that has given rise to no little discussion. Is the dementia (whatever the degree) the immediate result of the "nerve storm" of an epileptic attack, the exhaustional paralysis, so to speak, of the nerve cells - or is the demented condition part and parcel of the epilepsy as such. "The whole series of symptoms, bodily and mental,"[23] says Dr. Clouston, "may in some cases be the combined result of a general disturbance

of function or of disease in the brain, neither the convulsions being the primary disease, nor the mania, but both being equally the effects of the same cause." But may we not say that it is both cause and consequence, that these two things are so correlated as to move in a vicious circle, - that we have as a cause the hereditary disposition with its structural impairment and loss of integrity of the nervous elements in the cerebral cortex, and that as a consequence we get (when some element of stress is added) the epileptic attack? This, now acting as a cause what was formerly a consequence, still further impairs the nerve cells, and so the vicious circle is complete. The dementia comprising the interparoxysmal state may vary greatly in degree. It may manifest itself merely as a loss of memory, especially for recent events, or it may exhibit progressive grades of mental deterioration until we reach the last stage of all, the condition of terminal dementia. Most epileptics tend to become demented if they live long enough.

It may be asked, however, why, in so many cases, the fits continue for so many years, as they certainly do, and yet the patients may exhibit no great degree of dementia? The reason probably lies in the original structural condition of the nerve cells. We have seen that epilepsy is a disease "engrafted upon a cortical organic cellular anomaly which is induced largely by a faulty heredity, the exact

anatomic nature is not known." (vide supra) But it is not difficult to understand that this "faulty heredity" may not, and indeed is not, the same in degree (perhaps not in kind) in all cases. So that while, in one case, there may be a great degree of dementia induced, in another, with a different hereditary basis, a comparatively small degree of mental impairment results, even when the duration of the disease is the same. Again, the locality of the disease is to be taken into consideration in this question of mental impairment. It has already been stated that attacks which are purely psychic, or with slight spasm, leave behind them a more greatly impaired mental condition than those attacks which confine their manifestations mainly to the the motor side. The former have the seat of the lesion in the frontal lobes, the organs of the mind, consequently the degeneration of the nerve elements in this situation is more likely to result in the impairment of the mental faculties. Another point is that if the seizures commence at an early date, though the tendency is towards a long continuance of the disease, the mental state is not so impaired as might be expected, perhaps on the principle of adaptation, nature realising her threatened mental impotence, adapting other cells to carry on the work. As a clinical fact it is to be noted that when attacks occur in which severe motor convulsions and psychic phenomena are

combined, we have the severest type of the disease, the type least amenable to treatment, and the type most likely to terminate in complete dementia.

Another phase of the interparoxysmal state is the manio-depressive condition. This term is one used to designate those symptoms which are characteristic of alternate mania and melancholia. Both these types of insanity are exhibited by epileptics, and oftentimes in the same individual. Formerly it was more usual to speak of mania and melancholia as separate insane states, but it is now generally held that they are phases of one diseased process. So Krapelin in his lectures on Clinical Psychiatry, translated by Johnson, instead of making separate classifications of mania and melancholia, groups them under the one comprehensive term of manio-depressive insanity. The term, then, explains itself. Sometimes the one predominates, sometimes the other. In the interparoxysmal mania of epileptics we have an excited condition, both of motor and sensory functions, incoherence, exalted ideas and emotional states, while in the melancholia of epileptics we have a loss or diminution of psycho-motor activity, ideation is sluggish and the emotional state is characterised by depression. Both these conditions may be supplemented by delusions, hallucinations and (less frequently) by illusions. In the maniacal state the delusions are of an exalted or optimistic

character, while in the melancholic, they are of the suspicious or persecutory type.

Apart from actual maniacal or melancholic conditions, it is to be observed that the interparoxysmal state of the epileptic is one in which irritability, impulsiveness and religious emotionalism are very prominent. The brain cells are in a state of unstable equilibrium, and very little is required to produce an "explosion." Religious emotionalism is strongly developed. There is probably no class of cases in which the religious element enters so much into the thoughts, if not into the life, as the epileptic. Epileptic patients are very regular in attendance upon chapel services, though such services may not lessen their irritability, or influence their lack of inhibition. This feature - this religious tendency of epileptics - is it the consequence of the knowledge of their absolute helplessness, of the feeling that at any moment they may be stricken down, and encounter danger, meet with accident, or possibly death?

2. In the Pre-Paroxysmal states the maniacal epileptic is a most dangerous person, for at such times his psycho-motor activity is so intense and his muscular strength so enormous, his lack of responsibility so evident, that the damage he is able to do can hardly be over-estimated. Fortunately, these attacks are not of long duration, but while present they are rather alarming. The melancholic

condition is also more profound in the pre-paroxysmal state than in the condition between the attacks, the delusions are those of persecution and suspicion; and hallucinations (when present) are generally of a terrifying nature, such as wild beasts, snakes, etc. One would be inclined to characterise these hallucinations as visual aurae, and warnings of the coming storm.

With reference to the "automatism" as characterising the post-paroxysmal epileptic state, the subject is interesting from more than one point of view. It is interesting when one bears in mind the immunity from injury that such an "automaton" as a somnambulist has; it is interesting also from a medico-legal point of view, for while in this condition unlawful acts may be committed for which the patient is wholly irresponsible, and therefore ought not to be punished for that which he has done.

One sees with no little surprise the somnambulist walking down the stairs, not missing a step, making his way from room to room, and from floor to floor, locking and unlocking doors, passing backwards and forwards with eyes fixed and pupils dilated, but apparently seeing nothing; returning to bed, all unconscious of the acts committed, - and on awaking, by no effort of the memory is he able to recall the events of the period of somnambulism or automatism. It seems, therefore, impossible to carry the

memory of acts committed while in the automatic state out into the conscious state beyond. In post-epileptic automatism, amnesia is generally complete, and the question of responsibility of epileptics, not merely with regard to acts committed while in the post-paroxysmal automatic state, but generally, has a very important medico-legal bearing. Crimes are often committed during the maniacal phase of epileptic insanity - and as Clouston says:[24] "I have seen epileptic insanity take the form of a more acute maniacal condition than almost any other insanity, with the exception of mania from alcohol. There is no other form of insanity outside of asylums so frequently the cause of murders." And again: "Murder by an epileptic should usually be looked upon as being as much a symptom of his disease as larceny by a general paretic."

The following notes are from a case which illustrates the phase of post-epileptic automatism, and also brings clearly before us a number of the varied symptoms of epilepsy. I knew the patient very well, though she was not under my professional care, and I am indebted to my friend R. G. Tate, M.D., for these notes.

The patient was a girl of nineteen years of age, bright, intelligent-looking and of considerable conversational ability. Her attacks, sometimes few in number, one or two fits in twenty-four hours; sometimes many, sometimes

nine fits coming in rapid succession, were of a very severe nature, and came on, as a rule, at her menstrual periods, the great point of interest in them being the well-marked form in which each stage presented itself.

With regard to the presence of an aura, the only thing which forewarned her of an approaching fit was a feeling of intense cold coming over her face.

Let us take her attacks as they appeared to the onlooker. During a conversation, she would suddenly become silent, would yawn and grow deadly pale. Her eyes were fixed, gazing vacantly into space. She then fell to the ground, always on her left side, becoming at the same time perfectly rigid. The head was turned sharply to the left, and spasms began on the right side of her face, the mouth being drawn very much down on that side. The right arm, rigidly extended, was then swung slowly round from the shoulder over her head, at the same time coming from a position of supination to one of pronation. As it came to the front, it was slowly flexed, until at length it was close to the side, the hand being so acutely bent at the wrist that the fingers were thrust into the axilla. While this arm movement was in progress, the right leg was slowly drawn up and acutely bent at the knee. The tonic stage was followed by violent clonic spasms, chiefly of the trunk muscles, the body being bent sharply to the right side and

the strength of the contractions threw the patient about over the floor in a most marvellous manner. This stage lasted for a variable time, during which the patient performed various automatic movements. If one smiled at her, she smiled; if one frowned, she pouted her lips, wrinkled her forehead and frowned fiercely in return. She finally asked for water, which she gulped furiously until she had swallowed three to four pints of fluid. She then seemed to recover and cried, complaining of the pain of her arm, which was still tightly flexed, with the leg. The attack might be solitary or followed in rapid succession by others similar to it.

She seemed to be entirely unconscious from the time of the "aura" of cold until she had her last drink of water. The arm and leg which remained flexed were brought into their natural position with difficulty, and only then regained their power of natural movement. Whilst bringing the limbs into position, when the arm was nearly down, she picked up the mug from which she had just drunk in her left hand and immediately the right arm began to flex itself again and the hand to bend at the wrist against all efforts opposed to it.

It would almost appear that the action of the motor cells on the right side caused a stimulation through commissural action to the already over-excited cells on the

left side.

When the fits were coming on or passing off the patient fell into what Defendorf describes as the "dreamy condition" of epileptics, and during this time she showed very clearly how little epileptics are to be blamed for their actions even when they appear quite capable of controlling them. She appeared dazed and rarely spoke until she passed out of the condition, though as the doctor in charge said - "She would pass quite easily in a crowd as a normal individual." To take an example. One day when the doctor was watching her when so seized, she took his handkerchief, the end of which was hanging out of his pocket, and hid it in her dress. On coming to, she flatly denied the theft, and when the handkerchief was shown to her, she said it was her own. On its being pointed out that it smelt of tobacco, and hence could not belong to her, she replied:"O, yes, it is. At the last patients' dance I had no pocket in my dress, and Dr. T. put it into the pocket of his coat for me, and he smokes." Here we have the theft, and the ready reply of the kleptomaniac well exemplified. On another occasion she pulled the doctor's nose violently, although she at other times was on the best of terms with him. Taking acts like these into consideration,we may readily see how often the plea of temporary insanity may be advanced with truth as an extenuating circumstance in

the defence of a seeming criminal.

A curious fact with regard to this girl was that latterly she became subject to sudden attacks of motor aphasia, lasting sometimes for twenty-four hours, and during which she was perfectly conscious and able to express her thoughts in writing.

The family history of the patient was good. There had never been any serious disease in any relative. The patient had a fall at the age of two years, and bumped the back of her head. To this fact the epilepsy was attributed. She never had any severe illness or shock. The peculiar coarse features and irregular speech so commonly found in victims of epilepsy were entirely absent in this case.

I saw this patient in an attack on several occasions, and the peculiarities of the convulsive movements reminded one very forcibly of the cases of hystero-epilepsy described by Charcot. I sent the notes of the case to Sir W. R. Gowers who kindly gave his opinion as follows:-

" The above interesting case is clearly an example of true epilepsy followed by post-epileptic automatic and hysterical phenomena. The epilepsy is probably of the kind I have called "organic," resulting from an old stationary lesion of the brain in the left hemisphere, very likely a bruise on the surface resulting from the fall at two years of age. In women it is common for attacks of such simple

epilepsy during childhood to become associated with post-epileptic disturbance after puberty. The important thing is to recognise that the disease is essential epilepsy, and is to be treated as such, and not to be called 'hystero-epilepsy,' which was applied by Charcot to the more severe truly hysterical fits. Attacks are always essential, one or the other. The post-epileptic automatic actions are often curious. One patient always put into his pocket whatever was near him, and afterwards could not conceive how the object got there. Another man, a music-master, after attacks too slight to be noticed, began to take his clothes off, an action so equivocal when giving lessons to young ladies, that he had to discontinue his teaching work."

This case, then, is illustrative of several points already touched upon in the previous pages. The attacks had begun at an early age and continued with greater or less severity till the age of nineteen. There could not be said to be any degree of dementia. She was bright and intelligent. There was no facial expression such as is thought by some to be diagnostic of epilepsy. The clonic contractions comprised various kinds of movements, and the automatic actions of post-epileptic phenomena receive ample illustration. The patient lived nearly two years after the above notes were taken, and died of acute pneumonia.

Part VI.

Diagnosis and Prognosis.

The diagnosis of a case of the classic or grand mal type of epilepsy is hardly a matter of any difficulty, especially if the patient is seen throughout the course of the attack. Otherwise the physician is dependent upon the reports of friends, and these are often unreliable. The word "fit" is used very loosely in common parlance, referring to any attack from any cause in which unconsciousness is a symptom, so that it is necessary to question the friends in set and direct terms in order to arrive at a true significance of the facts of the case. The questions that should be asked are such as relate to the aura, the part of the body convulsed and the order of such convulsion, the presence of unconsciousness, the state of the patient after the cessation of the attack. It is when we come to the less frequent forms of epilepsy, the psychic form, and the epileptoid conditions, that errors in diagnosis are specially liable to be made. Let us, therefore, consider the chief conditions likely to be mistaken for epilepsy, and the first in importance is hysteria. It often happens that hysteria and epilepsy alternate in the same patient, and then the diagnosis is

still more confusing. The proper course for the medical man to take if in doubt is to withhold his final verdict until he has had an opportunity of seeing the patient in several seizures, not to trust himself to witnessing an isolated attack, and not to be guided entirely by the statements and opinions of friends. The chief points on which stress is to be laid in the differential diagnosis between hysteria and epilepsy are the existence of an aura, the presence of tongue-biting, the type of convulsions present, the duration of the seizure, the mental condition following an attack, the condition of the pupils, the state of the sphincters, the presence or absence of consciousness, and of automatism.

Difficulty sometimes arises in the diagnosis of syncopal attacks and attacks of petit mal without motor spasm. There is, however, generally some reason for the former, such as mental emotion, over exertion, heart disease or over-heated rooms. The suddenness both of attack and recovery is especially diagnostic of the epileptic seizure. Other points already noticed, namely, involuntary micturition and automatic movements, would, when present, confirm a diagnosis of epilepsy.

With regard to those conditions known as "epileptoid", due to organic brain disease, it is to be observed that the loss of consciousness, characteristic of epilepsy,

is rare, and that the history of the case would form an important guide.

General paralysis and epilepsy are liable to be confused only in the existence of the convulsions that are characteristic of certain periods of the disease, but by this time the other significant signs of general paresis are present, and by these it may be diagnosed. We pass over the question of simulation, considering it unnecessary to enter into details with regard to it. Most of the points already mentioned would be sufficient to give a clue to a correct diagnosis.

And here it may not be out of place to call attention to the condition known as the "facies epileptica." Aldred Turner says:[25] "So characteristic indeed is the facial expression of many epileptics that the term 'facies epileptica' has been applied to it." Spratling denies the existence of any special facial characteristic of epileptics, and says:[26] "The true facies epileptica, the only form possessing any distinctive worth from a diagnostic standpoint, is that made up of the trinity of results, namely, epileptic dementia, scars and bromic acne. These together produce a facial condition that cannot be mistaken, but being a composite picture of slow formation is of little or no value in the diagnosis of recent epilepsy."

Turner agrees so far, but says:"I do not think it can be denied that, in addition to the dulness and heaviness, combined with an emotional immobility, there is superadded a peculiar expression, difficult to define but readily recognised, which stamps the individual as an epileptic. I have seen the epileptic face associated with the slighter as well as the more profound degrees of dementia, and there is also a very small percentage of epileptics with normal mental attitude in whom this feature is present. I look upon the facies epileptica as a stigma of the degenerative neurosis producing the disease, of which convulsions and mental failure are further symptoms."

It is a pity Dr. Turner did not put into words a description of his facies epileptica. It may indeed be "difficult to define," but without a description of some sort, the ordinary observer is at a loss to know what to look for, inasmuch as the facies epileptica of which he makes mention cannot be considered an obtrusive phenomenon, as the 'facies' described by Spratling certainly is. I have not had the opportunity of seeing many cases of recent epilepsy, and those I have seen have made no impression upon my mind as having any special facial characteristics; the cases that come before me daily are those in which the mental faculties have become so

impaired as to necessitate their admission into an asylum, and therefore it is hardly possible for me to pronounce dogmatically upon the subject. One sees scores of cases which bear evidence of the "trinity of results" - the acne from continued use of bromides; the dull, heavy, demented countenance, the result of cerebral degeneration; and the scars on the scalp and supra-orbital ridges, the result of injuries; a combination bearing eloquent testimony that not only is the patient epileptic, but also that the disease has been long established. To avoid confusion with regard to the meaning of the phrase, however, it would, perhaps, be advantageous to confine the term "facies epileptica" to that facial phenomenon which according to Aldren Turner is one of the stigmata of the degenerative neurosis, and to use the phrases "facies bromica" and "facies post-epileptica" to indicate effects of bromism and traumata respectively.

As regards the question of prognosis, it may be said that epilepsy itself rarely causes death, patients may be subject to many fits per diem, and yet live for many years. When,however, status epilepticus supervenes there is always danger to life. Status epilepticus is the maximum development of epilepsy in which one paroxysm follows another so closely that the coma and exhaustion are continuous between seizures. It is in this condition

that the direct danger to life is greatest, the brain has no time to recover from the attacks, and each attack adds to the exhaustion present, till at length a point arrives at which life can no longer be prolonged.

Apart from this, danger to life arises from the liability to accidents. - Suffocation may occur during a seizure, or fatal burns may be contracted, or injuries from falling may have a fatal result.

But can epilepsy be cured, and if so, what is the percentage of cures effected? Since the introduction of bromides in the treatment of the disease, these questions can be answered favourably.

Aldren Turner states[27] that epilepsy can occasionally be cured. He studied 366 cases, and divided them according to the way in which they responded to treatment. Regarding as cured those cases in which the fits had been arrested for at least nine years, Turner arrives at the conclusion that 10.2 of epileptics are curable. Of the three types, the major attacks are more curable than the minor ones, these latter being the more intractable to treatment, and between the two come those cases in which the major and minor types were combined.

There are, however, several other factors to be taken into consideration with reference to the prognosis, in addition to the type of the disease, viz: the age at

which the disease commences, the time in relation to the onset of the disease at which treatment is first adopted, and the mental condition of the patient. The prognosis is bad when the mental condition is impaired, for in such cases the tendency is for this condition to become worse, often terminating in complete dementia.

Part VII.

The Treatment of Epilepsy.

In entering upon a consideration of the treatment of epilepsy, the first point of importance is what are the principles upon which treatment is to be carried out? We have a guide to our line of treatment of epilepsy, (and indeed the only true guide in the treatment of any diseased condition) in our knowledge (so far as it goes) of ~~the~~ its etiology and pathology. ~~of epilepsy~~. We have seen that epilepsy is an inherited disease, that its etio-pathology is most probably toxic or auto-toxic, that the immediate attack is due to an unstable equilibrium of the cellular elements, that the sensory elements which normally exert an inhibitory influence over the motor elements lose that authority and influence. With these findings fresh in our minds, it is not difficult to see that the rational treatment of epilepsy must be directed along broad and comprehensive lines; it must consist in endeavouring to overcome the effects of a degenerate heredity as far as possible, it must eliminate anything and everything that might be likely to produce a toxic condition of the blood, it must be devoted to increasing the general nutrition of the body (and thereby the nutrition of the cellular elements in the

cerebral cortex), it must be directed to a lessening of the activity of the motor elements, and to the removal of any conditions, centric or peripheral, which might be considered likely to prove a source of irritation.

It is convenient to consider the treatment of epilepsy under three headings, - general, medical and surgical. The general treatment refers to the treatment of the patient himself, and this is very important.

It is necessary that every care be taken to improve the patient's general bodily condition, and so of influencing those nutritional processes on which so much depends. The first great need of the epileptic is a sound and vigorous body. And in order that the patient may receive the maximum degree of benefit from general treatment it is obvious that he should submit himself to the care and control of others, and in so doing exercise his own self-control. Self-control is an important element in the moral treatment of epilepsy, as indeed it is in all other morbid conditions of the nervous centres. Nowadays the chronic epileptic is isolated from his home surroundings, which oftentimes are not very conducive to health, and is placed under such hygienic conditions as have been found most suitable for the treatment of this disease. "Colonies" have been established in various places, at which the patient is taught to employ himself in simple

or complex vocations, his own peculiar personal needs are provided for, amusement and recreation go hand in hand with toil and labour, his diet is decided upon and apportioned with a view to his special necessities, his freedom is almost perfect, his fresh air is umlimited, he lives a systematic outdoor life,& in short, he is made to feel that he is "at home from home."

With regard to the important consideration of diet, the first question that naturally occurs to one is - What is the principle which underlies the dietetic treatment of epileptic fits? Is it not that sometimes the fit is the result of a paroxysmal toxoemia, and that the toxic element is introduced by the ingesta and either formed in the digestive tract or else is the result of normal metabolism? We do not know the precise nature of this toxic element, and therefore dietetic experiments are more or less empirical. Many modifications in diet have been tried, and as a result, certain conclusions have been arrived at. It has, for instance, been long considered by competent authorities that meat is harmful in epilepsy, and that, on this account, it should either be omitted from the dietary or the quantity strictly limited. It would appear, however, from experiments made by Dr. A. J. Rosanoff, of New York, and reported in the Journal of Nervous and Mental Disease for December 1905, that the

cause of the convulsions is not meat as such, or any particular kind of food, but the absolute quantity of proteid matter irrespective of its origin. Eleven patients suffering from long standing epilepsy were kept for prolonged periods on five different diets, being confined to bed and receiving no medicine. The first observation was made with an ordinary mixed diet in which the amount of proteid was small, that of the carbohydrates large, and of the fats moderate. In the second case a vegetable diet was given which differed from the previous one only in the substitution of vegetable for animal proteid, the quantities of the proximate principles being practically the same in both. The results in these two instances were practically alike as regards the mental condition exhibited by the patients, the maintenance of weight and the number of convulsions per week. So That the effect of a mixed diet in epilepsy does not differ from that of a vegetable diet containing the same quantities of proximate principles.

In the third and fourth cases the features of the diet were respectively an insufficient and an excessive amount of proteids. In both cases the patients were restless and irritable, and in both the number of convulsions per week rose in about equal degree. If therefore the amount of proteid is either below or above the

indispensable minimum the severity of the disease is increased. In the fifth instance the diet contained a large excess of proteid and was very deficient in carbo-hydrates. The result was a general aggravation of the mental and physical disturbances exhibited by the patients, who became dangerous, noisy, restless and destructive, and mostly lost weight, whilst the convulsions greatly increased in number per week. This result is ascribed to the compulsory use by the organism of proteid in place of carbo-hydrate material. Carbo-hydrates and fats, it is pointed out, are to a certain extent capable of replacing proteids in the diet, and this with advantage in the case of the epileptic, who should therefore receive the largest amount of these principles that he can assimilate without inconvenience and the smallest amount of proteid which is compatible with the preservation of nitrogenous equilibrium, the nitrogen ingested not being allowed to fall below the nitrogen excreted.

There seems to be no doubt that excessive consumption of proteid results in the circulation, in the blood, of imperfectly oxidised nitrogenous matters. The same thing will happen if the carbo-hydrates are consumed in excess, for then the complete oxidation of the proteids is interfered with, so that in either case there is an alteration in the quality of the blood, which may at length result in toxoemia.

Dr. Aldren Turner in the Practitioner for April 1906 reports favourably of the influence of a diet free from purin bodies, (substances constructed on a base C5N4) and selected his dietary from the following articles of food - milk, eggs, butter, cheese, rice, macaroni, tapioca, white bread, cabbage, lettuce, cauliflower, sugar and fruit; avoiding tea, coffee and cocoa, and all kinds of fish, fowl and meat (including sweetbread).

The suggestion of Toulouse and Richet that a salt-free or salt-limited diet would be advantageous, ought not to be omitted in this connection. It was thought that by diminishing the quantity of chlorides a less quantity of the bromide salt would suffice, and those who have tried this plan speak of it as being undoubtedly beneficial.

Other considerations of importance are necessary to be observed, viz: regularity of meals, moderation in quantity, and thoroughness in mastication. Under these conditions the liability to gastro-intestinal disorders, which we have already considered among the exciting causes of the disease, would be reduced to a minimum.

Passing on to the medicinal treatment, we ask first, what can we do to <u>prevent</u> an attack of epilepsy. And here comes in the value and importance of the aura, when present. For if the patient is cognisant of an approaching attack, he can then take steps in the hope of

arresting the threatened danger. One of the drugs used for this purpose is nitrate of amyl. Glass cachets containing a few minims of the drug are carried in the pocket, and one is broken in a handkerchief and the vapour inhaled. Probably the reason for the beneficial action of this drug lies in the fact that it is a vaso-dilator, and, according to Gowers, "floods the brain with arterial blood, a potent agent for modifying the action of nerve elements."

If the aura is felt in arm or leg, arrest of the fit has been secured by tying a ligature tightly round the affected limb. This method is an old one, and is mentioned by Sir Thomas Watson in his clinical lectures, Vol. (p.665). A full dose of bromide, the patient lying down meanwhile, is also successful oftentimes in warding off an attack.

When, however, the fit is inevitable, little can be done beyond preventing the patient injuring himself. The clothing round the neck and chest should be loosened to prevent cerebral engorgement and interference with respiration.

With regard to the drugs used to arrest the attacks or to diminish their severity, the chief place must be given to the bromides. They were introduced by Locock in 1857, and still hold their own in the therapy of epilepsy. The salt almost universally used is the potassium salt,

but the ammonium and sodium salts have practically the same physiological action. Bromides diminish the irritability of the cortex. They act upon the sensory elements of the cortex. They reduce (according to Prout and Clark) the intensity of afferent impulses by the motor cells, and in this way aid cerebral inhibition and lessen the liability of the occurrence of a nerve storm. In addition to the production of an acneiform eruption, the long continued use of the bromides in large doses is sometimes attended with disastrous results to the nervous system in that they produce a stuporose effect upon the patient and cause a blunting of the mental faculties. This constitutes the great drawback to their prolonged administration.

The digestive function is also impaired by prolonged bromide administration, the normal secretion of the gastric juice is checked, causing nausea and diarrhoea. Other untoward consequences are unsteadiness of gait, impotence, amnesia and aphasia. To obviate, as far as possible, these unfortunate results, several points deserve consideration. The drug should be administered at first in small doses, and the first toxic effects noted. The intestinal canal should be kept in good condition, and the prevention of toxicity promoted by the occasional exhibition of such intestinal antiseptics as salol and ichthyol. Constipation should be carefully guarded against.

The acne so common in connection with the use of bromides can be diminished or prevented by the administration of Liquor Arsenicalis in conjunction with the salt.

It has been already stated that a less dose of the bromide is sufficient if the patient be fed on a salt-free diet. This is, of course, sometimes difficult in practice, especially in the case of private patients, but the suggestion is worth trial, as it has been estimated that half the quantity of bromide is sufficient, and of course the liability to bromide intoxication would be thus greatly lessened.

We have seen that the bromides act as a depressant to the nervous system, that they aid the inhibition of the sensory elements of the cerebral cortex; but there is another point connected with their therapeutic action which is worthy of notice. There can be no doubt that the bromides act by virtue of the element bromine which they contain, and though it is not certainly known how this element affects the nerve cells, it is by no means improbable, seeing that bromine is a powerful disinfectant, that this element neutralises toxic elements in the blood, and thus lessens the metabolic changes of degeneration.

The administration of opium with bromide was recommended by Fleschig in 1893. The idea was to prepare the system for the action of the bromides by small but

increasing doses of opium for about six weeks, and then suddenly withdrawing it, and exhibiting large but decreasing doses of the bromide salt. This plan has not met with much favour by those who have given it a trial, and is now almost entirely given up.

Sir W. Gowers introduced borax as a substitute for the bromides, but it does not seem to have any greater influence over the disease than the bromides have, while its use is liable to be attended with gastro-intestinal and cutaneous disorders. Many other drugs have been used with varying success, among which may be mentioned chloral hydrate (especially useful in status epilepticus when combined with bromides), nitro-glycerine (used in epileptoid attacks associated with arterio-sclerosis), zinc and iron. Salicylate of soda has also been suggested for the treatment of epilepsy. This suggestion has been made on Haig's theory of the excess of uric acid in the blood. Allusion might be made to Ceni's serum treatment, which was based on his belief in the existence of a biochemical toxic substance in the blood of epileptics. It has not been proved to be of any great practical value.

Iron is useful in those cases where epilepsy is associated with anaemia, but beyond its value as a haematinic, it probably has no direct effect upon the

disease. Many other remedies have been suggested by pharmacologists, and while we should not forget the necessity for the removal of all possible causes of peripheral irritation, such as errors of refraction, etc., yet we must not allow the bromides to fall into disuse but regard them as our sheet anchor in this intractable complaint.

And now in what class of cases may the bromides be expected to have the best effect? They are undoubtedly of most use ~~with~~ in the classic type of epilepsy, those cases of idiopathic 'grand mal' in which the convulsive element is predominant; they succeed next best in the cases of petit mal with minor spasm; while in the true petit mal or in psychic epilepsy, as also in the traumatic form, they are of little value.

In the administration of bromides, attention should be paid to the time when the drug should be given. Seeing that it has a direct sedative effect upon the cerebral nervous system it follows that the most suitable time for its administration is shortly before the patient may be expected to have an attack. If a faithful record be kept of all seizures, it will, in many cases, be easy to determine the time when attacks are the more likely to occur, and the medicine should be administered at these times. If, for instance, the attacks occur most frequently during the night, the medicine should be given before retiring, or if

they occur generally in the early morning, a large dose should be given before the patient rises.

The surgical treatment of epilepsy may be dismissed with a brief reference, as the results obtained are on the whole unfavourable. The two classes of cases in which surgical treatment has been requisitioned are those of grand mal and Jacksonian epilepsy. Of the former, only those cases where trauma is considered the exciting cause, can be judged suitable for operative interference. Idiopathic epilepsy and epileptoid conditions due to organic disease stand outside the pale of such treatment. Spratling gives[28] a very interesting series of cases operated upon for relief of epilepsy, and says that the majority of those operated on experienced no benefit, while about a fourth were benefited somewhat.

Little can be added as to the treatment of epileptic insanity. It is simply that of epilepsy with that of mania superadded. In these cases the prognosis is not so good. The disease is often too far advanced to hope for a cure; the most that can then be looked for is a diminution in the severity of the symptoms by the exhibition of sufficiently large doses of the bromides; but, as we have seen, large doses of the drug have disastrous effects. It adds to the mental enfeeblement, and with both these forces fighting against him in combination, namely, the

attacks of epilepsy and the drugs given for their alleviation, little wonder is it that the patient's path is downwards, his mental degeneration increases, and in a comparatively short space of time he is reduced to that sad and sorrowful state of dementia which is so common a sight, albeit so pitiable, in the epileptic wards of our county asylums. It is hardly necessary to refer to the treatment of this state, which is really one of careful nursing, or to the treatment of the interparoxysmal condition, when the usual sedatives and hypnotics may be exhibited when necessary to combat the hyper-excitation of the patient.

Epilepsy is an intractable disease. It has long baffled medical skill. In olden times it was a disease associated with the supernatural, and enshrouded in mystery, and though we have (so we think) eliminated the supernatural, yet it does not appear that we are much nearer the solution of the mystery. The structural disposition of the inherited abnormal nerve cells, the nature of the excitant which issues in the production of the convulsion, the mechanism by which conscious life is suddenly held in abeyance and as suddenly restored; these form, so to speak, a triple veil hiding from view the mystery of the epileptic phenomena, for the penetration of which we must await future researches in the threefold realm of histopathology, bio-chemistry and psychology.

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